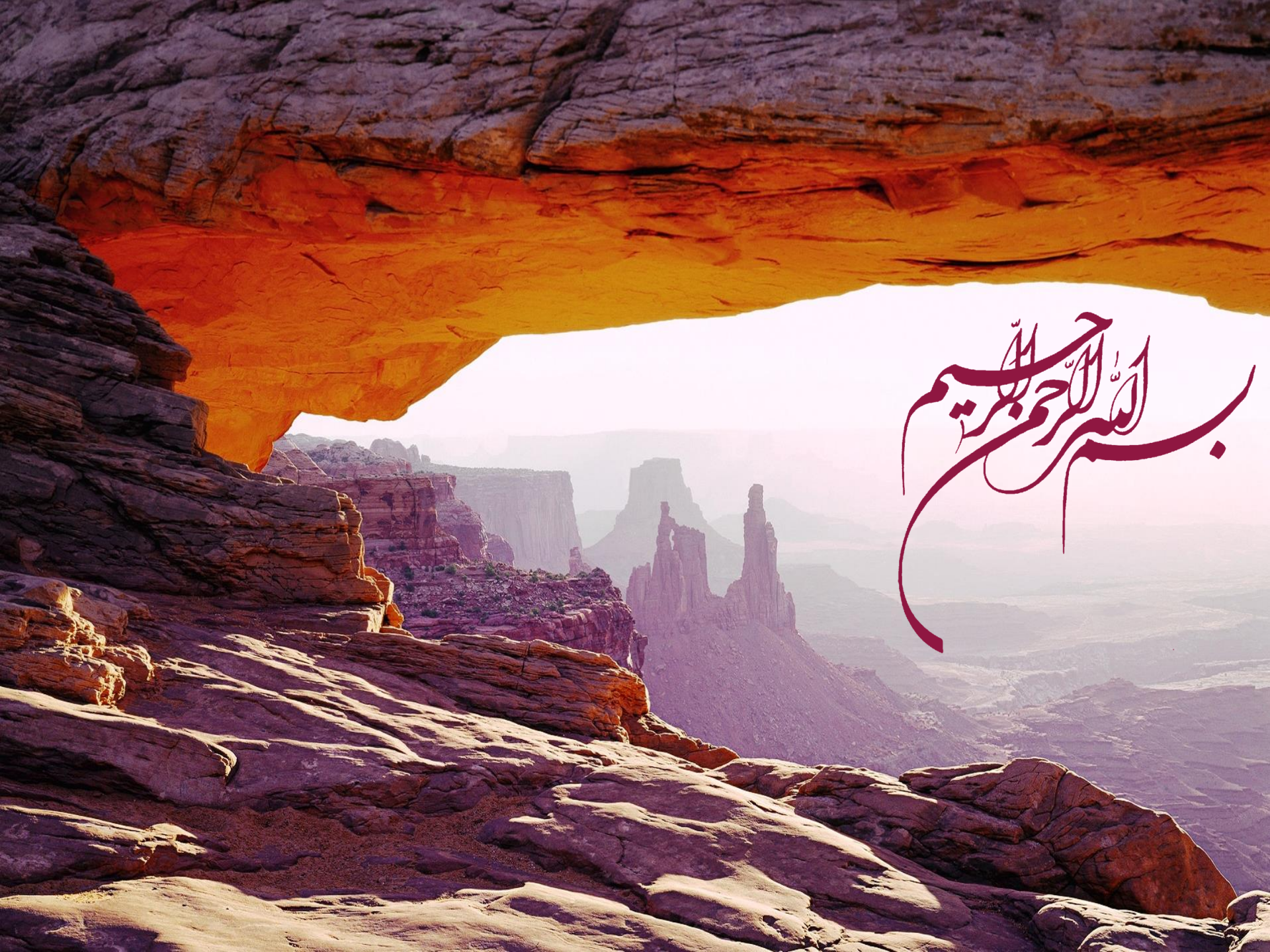

بسم الله الرحمن الرحيم

UMBILICAL CORD BLOOD ANALYSIS

- **Umbilical cord blood analysis for evaluation of the newborn's acid-base status immediately after delivery is the most objective way of assessing the fetal metabolic condition at birth .**

- **This information can also be useful from medical and medicolegal perspectives since it excludes perinatal asphyxia or hypoxia secondary to the birth process**

- A normal umbilical artery blood pH virtually excludes "birth asphyxia."

INDICATIONS:

- There is no consensus regarding indications for umbilical cord blood acid-base analysis postdelivery.
  - ACOG recommends that cord blood gas and pH analyses be obtained in the following circumstances:
    - Severe IUGR
    - multifetal gestations
    - intrapartum fever
    - maternal thyroid disease
    - breech deliveries
    - preterm births
    - meconium staining
    - abnormal FHR pattern(category II,III)
    - low Apgar scores(<5at5 minutesand10minutes)

- The Royal College of Obstetricians and Gynecologists and the Royal College of Midwives, recommend that cord blood acid-base analysis be "considered" for all deliveries

- only 30 to 40 % of newborns who are depressed (ie, have low Apgars) at birth are acidotic at delivery, which suggests that the depression is related to factors other than prolonged hypoxia

- The pH, PCO_2, PO_2, hemoglobin, Bicarbonate concentration,oxygen saturation, and base excess (or deficit) can be measured.
- The most useful values for interpretation of fetal-newborn condition and prognosis are the pH and base excess (or deficit) .

فرمول HENDERSON-HASSELBAKH

$$pH = pK + \log \frac{[base]}{[acid]} \quad \text{or,} \quad pH = pK + \log \frac{HCO_3^-}{H_2CO_3}$$

پارامتر pk در این فرمول ضریب ثابت تجزیه است و برای هر سیستم بافری عدد خاصی است که در مورد سیستم اسیدکربیک- بیکربنات این ضریب 6/1 خواهد بود

- For clinical purposes, HCO_3^- represents the metabolic component and is reported in mEq/L.
- The H_2CO_3 concentration represents the respiratory component and is reported as the PCO_2 in mm Hg.

با توجه به روند تبدیل CO_2 به اسیدکربنیک وسپس یون بیکربنات میتوان چنین نوشت

$$CO_2 + H2O \rightarrow H_2CO_3 \rightarrow H + HCO_3^-$$

$$\uparrow CO_2 \rightarrow \uparrow HCO_3^-$$

$$\downarrow CO_2 \rightarrow \downarrow HCO_3^-$$

پس فرمول هندرسون- هسلباخ بصورت زیر درمی آید:

$$pH = pK + \log \frac{\text{metabolic } (HCO_3^- \text{ mEq/L})}{\text{respiratory } (PCO_2 \text{ mmHg})}$$

Every 1meq/l of Hco3 - increases the PH by 0.1

Every 10 mmHg of Pco2 reduces the PH by 0.08

(BE) Base dificit, Base Excess

با توجه به روند تبدیل co_2 به اسیدکربنیک وسپس یون بیکربنات میتوان چنین نوشت

$$co_2 + H2o \rightarrow H_2co_3 \rightarrow H + Hco_3^-$$

$$\uparrow co_2 \rightarrow \uparrow Hco_3^-$$

$$\downarrow co_2 \rightarrow \downarrow Hco_3^-$$

بنابراین میزان یون بیکربنات در راستای تغییر co2 دستخوش تغییر خواهد شد. به عبارت دیگر Hco_3^- به راحتی تحت تاثیر تغییرا ت تنفسی قرار میگیرد. بنابراین با این توصیف نمی توانیم Hco_3^- را بعنوان یک پایه محکم برای قضاوت بازوی متابولیک تعادل اسید و باز مورد استفاده قرار دهیم.

بمنظور این قضاوت نیاز به پایگاه مستحکم تری است و آن Base dificit و Base Excess است ، در عمل هردو پارامتر را بصورت BE ولی با علامت مثبت یا منفی نشان میدهند.

- Base deficit does not significantly change during respiratory acidosis.

اسیدوز تنفسی به حالتی اطلاق می شود که فشار co2 شریانی از مرز طبیعی 50 mmhg افزایش یابد.

$PH \downarrow$

$Pco_2 \uparrow$

$Hco_3^- \uparrow$

$BE \uparrow +$

اسیدوز متابولیک در اثر کاهش Hco3 - یا اضافه شدن یون هیدروژن ایجاد میشود.

$PH \downarrow$

$Pco_2 \downarrow$

$Hco_3^- \downarrow$

$BE \downarrow$

$Paco_2 \downarrow$

- In the fetus, the placenta serves as both lungs and to kidneys.
- One principal cause of developing fetal acidemia is a decrease in uteroplacental perfusion. This results in the retention of CO2 (respiratory acidemia) and a mixed or metabolic acidemia develops when oxygen deprivation is of sufficient duration and magnitude to require anaerobic metabolism

- In fetus, respiratory and metabolic acidemia, and ultimately tissue acidosis, are most likely part of a progressively worsening continuum.

- This is different from adult pathophysiology, in which distinct conditions result in either respirator or metabolic acidemia.

- Fetal oxygenation and pH generally decline during the course of normal labor.

- Most fetuses will tolerate intrapartum acidemia with a pH as low as 7 without incurring neurological impairment.

- the duration of neonatal metabolic acidosis after delivery is also an important prognostic factor.

NORMAL VALUES:

- The mean umbilical blood pH and gas values for premature and term infants are almost identical, although the premature infant may have low Apgar scores because of prematurity itself.

Range of mean umbilical artery blood pH and gas values in preterm newborns

- pH 7.25 to 7.29
- PCO_2 (mmHg) 49.2 to 51.6
- HCO_3-(mEq/L) 22.4 to 23.9
- Base excess (mEq/L) -2.5 to -3.3

Range of mean umbilical artery blood pH and gas values in term newborns

- pH 7.27 to 7.28
- PCO_2 (mmHg) 49.2 to 50.3
- HCO_3-(mEq/L) 22.0 to 23.1
- Base excess (mEq/L) -2.7 to -3.6

- the American Academy of Pediatrics and the American College of Obstetricians and Gynecologists, defined metabolic acidosis as umbilical arterial pH <7 and a

  BE of at least 12 mmol/L.

NOTICE

- ***Respiratory acidemia*** generally develops as a result of an acute interruption in placental gas exchange with subsequent CO2 retention.
- Transient umbilical cord compression is the most common antecedent factor.
- respiratory acidemia is not harmful to the fetus.
- The degree to which pH is affected by PCO2, the respiratory component of the acidosis, can be calculated.

- First, the upper normal neonatal PCO2 (49 mm Hg) is subtracted from the cord blood gas PCO2 value.
- Each 10 additional mm Hg PCO2 will lower the pH by 0.08 units. Thus, in a mixed respiratory-metabolic acidemia, the benign respiratory component can be calculated .
- example: During labor, an acute cord prolapse occurred and the fetus was delivered by cesarean 20 minutes later. The umbilical artery blood gas pH was 6.95, with a PCO2 of 89 mm Hg. To calculate the degree to which the cord compression and subsequent impairment of CO2 exchange affected the pH, the relationship given earlier is applied: 89 mm Hg minus 49 mm Hg = 40 mm Hg (excess CO2). To correct pH: (40 ÷ 10) x 0.08 = 0.32; 6.95 + 0.32 = 7.27. Therefore, the pH prior to cord prolapse was approximately 7.27, well within normal limits. Thus, the entire pH resulted from respiratory acidosis.

ANOTHER EXAMPLE:

A patient admitted with a nonreassuring fHR tracing and rushed for C/S

Cord blood gas: PH=6.90 Pco2 =60mmhg

60-50=10

Every 10 mmHg of Pco2 reduces the PH by 0.08

10 ÷10 = 1

1 × 0.08 = 0.08

6.90 + 0.08 = 6.98 :a level of damaging acidosis

The cord blood gases, uncorrected by respiratory component and cannot support a recent asphyxia event.

In fact they cannot tell us when the damaging acidemia occurred

- When metabolic acidosis is found , the timing of hypoxic insult cannot be estimated .
- In contrast, when the respiratory component is found, the onset of hypoxic insult can be established because this component cannot last more than 20-30 minutes.
- Furthermore one can establish what the PH was before the acute insult by reducing the respiratory component .

- Umbilical artery PO2 is not predictive of any adverse neonatal outcome.

- Respiratory acidosis alone is not usually associated with complications in the newborn

- Newborns with hypoxia proximate to delivery that might result in subsequent neurologic injury should manifest the following :
  - Umbilical artery blood pH < 7
  - A metabolic component
  - Apgar scores of ≤ 3 for > 5 minutes
  - Seizures, coma, hypotonia
  - Evidence of multisystem organ dysfunction in neonatal period

LIMITATIONS OF FETAL BLOOD GAS ANALYSIS

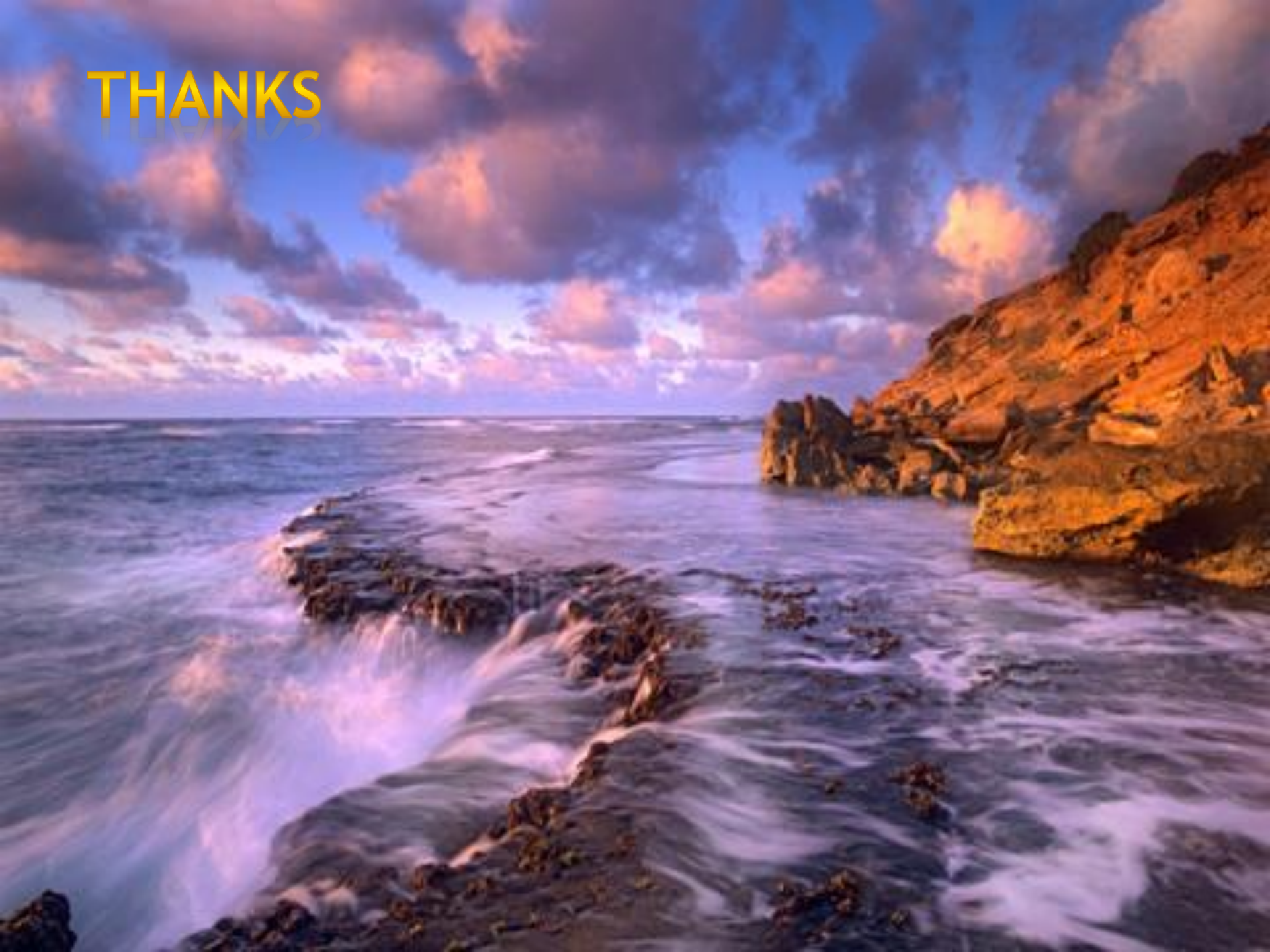
- **cord blood pH value alone does not distinguish between a primary fetal or placental disorder and the indirect effect of a maternal acid-base disorder.**
- Since fetal pH is normally 0.1 unit lower than that of the mother, maternal pH should be determined if fetal acidosis is suspected to be a passive result of maternal acidosis.
- **Fetal blood gases also do not necessarily reflect asphyxia occurred remote from delivery .**
- **Co-morbidities (eg, fetal growth restriction,anemia) are important biological modifiers of neurological and other end-organ risk.**

TECHNIQUE :

- Blood collection is performed following delivery by immediately isolating a 10- to 20-cm segment of cord with two clamps near the neonate and two clamps nearer the placenta ,Because delay of 20 to 30 seconds can alter both the PCO2 and pH .
- The cord is then cut between the two proximal and two distal clamps.
- Arterial blood is drawn from the isolated segment of cord into a 1- to 2-ml plastic syringe that has been flushed with a heparin solution containing 1000 U/mL. The needle is capped and the syringe transported, on ice, to the laboratory.

- cord blood sample prepared in this way is reasonably stable for assessment of both pH and base deficit for 60 minutes. Ideally, the test is performed as soon as possible after delivery.
- Arterial pH and base excess at birth can be estimated from testing a blood sample obtained from a clamped umbilical cord kept at room temperature up to 90 minutes after birth.

- If difficulty is encountered in obtaining umbilical cord artery blood samples, the vessels on the fetal surface of the placenta can be utilized ("arteries cross over veins") and will provide similar, but not necessarily equivalent, results .
- Some clinicians prefer to obtain a sample from both the artery and vein, although the cost-effectiveness for this practice has not been established .
- If both vessels are sampled, the median arteriovenous pH difference is 0.09 (range 0.02-0.49).


THANKS